Oculomotor Nerve Palsy Caused by Posterior Communicating Artery Aneurysm: Evaluation of Symptoms after Endovascular Treatment

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Summary

We report the outcome of endovascular treatment in a series of patients presenting with posterior communicating artery aneurysm causing ocular motor nerve palsy. A retrospective study was made of ten patients who were treated by coil embolization of posterior communicating artery aneurysm caused by oculomotor nerve palsy. The assessed parameters were as follows: patient's age, presence of subarachnoid hemorrhage, aneurysm size, preoperative severity of symptoms, and timing of treatment after onset of symptoms. Improvement of oculomotor nerve palsy after treatment was noted in eight patients (80.0%). Complete recovery was noted in seven patients (70.0%), partial recovery in one patient (10.0%), and no recovery in two patients (20%). Clinical presentations with early management (≤2 days) were significant in influencing recovery. Complete recovery from ocular motor nerve palsy was significantly higher in patients with initial incomplete palsy compared with initial complete palsy patients (6/6 versus 1/4). Early treatment and initial partial palsy are relevant to improving prognoses. Endovascular treatment is favored method for treating oculomotor palsy.

Introduction

Since the 1990s, endovascular coil embolization, rather than microsurgical clipping, has become the primary alternative treatment for treating intracranial aneurysms. Certain cerebrovascular diseases, such as cranial neuropathy, are caused by conditions such as the aneurysm. Nonetheless, the effectiveness of endovascular treatment of cerebrovascular diseases with the mass effect has been questionable. This is because coiling does not resolve all of the mass effect of an aneurysm, and the coil mass itself could contribute to the mass effect.

However, recovery from cranial nerve palsy after endovascular coiling on cerebral aneurysms has recently been documented [1-4]. The reports consider the loss or decrease of arterial pulsation afforded by coil embolization to play an important role in recovery from oculomotor nerve palsy. It is true that surgical decompression to reduce the mass effect is not a unique method for improving the symptoms of cranial nerve palsy. In this paper, we report on the follow-up results for ten cases of cerebral aneurysms treated by endovascular coiling, and review the relevant literature.

Material and Methods

A retrospective study was made of ten patients treated by coil embolization of a posterior communicating artery (Pcom) aneurysm caused by oculomotor nerve palsy between January 2004 and May 2010. Medical and radiological data were reviewed in all patients. The demographic data of patients are shown in Table 1. There were one man and nine women with a mean age of 60 years (range from 38 to 84 years). Nine cases were ruptured PCom an-

eurysms, one was an unruptured aneurysm. All patients with ruptured aneurysm were admitted at the time of bleeding and were treated within 24 hours after admission. Information on time of initiation of oculomotor nerve palsy was obtained from patients or their relatives. Severity of symptoms was checked at admission. The assessed parameters were as follows: patient's age, presence of subarachnoid hemorrhage, aneurysm size, preoperative severity of symptoms, and timing of treatment after onset of symptoms. The specific details of each parameter were as follows: age was under 55 years or over 55 years, aneurysm size was either less than or equal to 10 mm or was greater than 10 mm, and timing of treatment was within two days or after two days.

Complete oculomotor nerve palsy was defined as patients with all symptoms, such as ptosis, mydriasis, and diplopia. Partial oculomotor nerve palsy was defined as patients with one or two of the above symptoms. Patients' recovery from oculomotor nerve palsy after treatment was classified as follows: no recovery if all three symptoms persisted (ptosis, mydriasis, and diplopia), partial recovery if one or two of the three symptoms persisted, and complete recovery if no symptoms of oculomotor nerve palsy were present.

Because all patients were treated within 24 hours after admission, they were not pretreated with oral antiplatelet therapy for several days. All procedures were performed under intravenous sedation using by Propofol and Alfentanyl. At the beginning of the procedure, a patient was pretreated with a heparin bolus (5000 IU) injection, followed by 1000 IU/hour of heparin. The patient was heparinized to an activated clotting time (ACT) of 250 to 300 seconds during the procedure. In stent assisted coiling cases, intravenous Aspirin Lysine® (900 mg aspirin) was injected immediately after stenting. And dual antiplatelet therapy (75 mg clopidogrel daily for two weeks and 100 mg aspirin daily for four weeks) was given after the procedure. The coil embolization was finished when blood flow into the aneurysm was not shown in the conventional angiogram. Overall, clinical follow-up ranged from two to 18 months, with a mean period of 12 months, and angiographic follow-up was performed at six months and 12 months after discharging patients from the hospital. The recovery state of oculomotor nerve palsy was assessed by an ophthalmologist.

Results

The results of the patients and the factors correlated with recovery are shown in Tables 1 and 2. Improvement of oculomotor nerve palsy after treatment was noted in eight patients (80%). Complete recovery was noted in seven patients (70%), partial recovery in one patient (10%), and no recovery in two patients (20%). Six out of seven patients with complete recovery had pre-operative partial oculomotor nerve palsy. Patients who experienced the most complete recovery encountered improvement of mydriasis within two days to three months after treatment. Improvement of diplopia took longer and occurred within 18 months. In one partial recovery cases, one patient had partially improved diplopia and complete recovery from mydriasis and ptosis.

The timing of treatment after the onset of symptoms was one to two days in five patients, three to 14 days in three patients, and more than 14 days in two patients. Time to recovery after treatment was one to four weeks in three patients and more than four weeks in five patients.

The one partial recovery case had complete nerve palsy and an unruptured large aneurysm (19.0×16.0 mm). In the two patients who experienced no recovery of symptoms, both had complete nerve palsy and were treated much later (>20 days) than those who experienced recovery.

All six patients who had partial cranial nerve palsy initially experienced complete recovery, and wo out of four who had complete cranial nerve palsy experienced complete recovery in contrast (100 Vs 50.0%). All five patients who were treated within two days after onset of symptom experienced complete recovery, and only two of the five patients who were treated after two days completely recovered (100 Vs 40.0%). We consider that complete recovery of symptom is more likely with partial oculomotor nerve palsy than complete palsy patients initially and early management may influence recovery.

Discussion

About one third of PCom aneurysms may induce oculomotor nerve palsy because of their anatomical proximity to the oculomotor nerve [5]. Aneurysms located in the cavernous ICA are

Table 1 **Demographic data of patients.**

No	Age/ Sex	Location	SAH	Size (mm)	Third nerve palsy	Recovery status	Timing of treatment (days)	Time to recovery (weeks)
1	62/F	Rt. PCom	SAH	9.7 X 5.9	PP	CR	2	2
2	65/F	Rt. PCom	SAH	7.0 X 4.0	PP	CR	2	6
3	66/M	Rt. PCom	SAH	5.3 X 3.3	CP	CR	1	1
4	66/F	Lt. PCom	SAH	6.6 X 4.4	PP	CR	3	10
5	38/F	Rt. PCom	SAH	6.9 X 2.1	PP	CR	5	8
6	56/F	Lt. PCom	no	16.0 X 19.0	CP	PR	3	12
7	44/F	Rt. PCom	SAH	26.8 X 22.0	PP	CR	2	10
8	69/F	Rt. PCom	SAH	9.2 X 3.4	CP	No	30	–
9	84/F	Rt. PCom	SAH	5.2 X 6.4	CP	No	20	–
10	52/F	Rt. PCom	SAH	6.5 X 2.0	PP	CR	2	2

An: aneurysm
ICA: internal carotid artery, T-CCF: traumatic carotid cavernous fistula, PCom: posterior communicating artery.
CP: complete palsy, PP: partial palsy
CR: complete recovery, P: partial palsy.

Table 2 **Relationship of recovery and factors correlated with the degree of nerve recovery**

Factor	Cases(N)	CR(%)	PR(%)	NC(%)
Age				
< 55 years old	3	3(100)	0(0)	0(0)
≥ 55 years old	7	4(57.1)	1(14.3)	2(28.6)
Presence of SAH				
SAH	9	7(77.8)	0(0)	2(22.2)
No SAH	1	0(0)	1(100)	0(0)
Aneurysm size				
< 10 mm	8	6(75.0)	0(0)	2(25.0)
≥ 10 mm	2	1(50.0)	1(50.0)	0(0)
Severity of symptom				
Partial	6	6(100)	0(0)	0(0)
Complete	4	1(25.0)	1(25.0)	2(50.0)
Timing of treatment				
≤ 2 days	5	5(100.0)	0(0)	0(0)
> 2 days	5	2(40.0)	1(20.0)	2(40.0)
Total	10	7(70.0)	1(10.0)	2(20.0)

CR: complete recovery, PR: partial recovery, NC: no change.

known to cause a variety of cranial nerve dysfunction including III, IV, V, and VI [6,7]. In some cases, the presenting symptoms may vary according to the location and direction of the aneurysm in relation to the course of the cranial nerve within the sinus. Additionally, aneurysms leading to oculomotor nerve palsy are located in the top of the basilar artery, in the superior cerebellar artery and in the anterior choroidal artery [8-10]. Because isolated third nerve palsy caused by an intracranial aneurysm usually heralds impending rupture of the aneurysm [2], urgent treatment is required.

There are various theories for the mechanism of oculomotor nerve palsy caused by aneurysm. Those theories posit the following mechanisms: direct mechanical compression of the oculomotor nerve by enlargement of the aneurysm in the suprasellar cistern, irritation of subarachnoid hemorrhage and nerve injury by arterial pulsation of the aneurysm (called the "water hammer effect") and by pressure from arterial bleeding due to rupture of the aneurysm [1,2,4,11].

To date, microsurgical clipping has been considered the treatment of choice for aneurysms that lead to cranial nerve symptoms, because the main mechanism of cranial nerve palsy has been considered to be the mechanical mass effect of the intracranial aneurysm. However, there are recently reported cases in which the oculomotor nerve palsy was resolved after coil embolization of PCom aneurysms [1-4]. Birchall et al. [1] reported complete recovery from symptoms after coil embolization in three patients with PCom aneurysm–related oculomotor nerve palsy. Also, Mavilio et al. [4] presented similar results in six patients.

They considered the disappearance of aneurysmal pulsation after coiling to be the main mechanism for the recovery from oculomotor nerve palsy. Unlike microsurgical aneurysm clipping, endovascular treatment with coils does not remove the mass effect of an aneurysm immediately. However, the loss or decrease of arterial pulsation afforded by coil embolization may be more important for early resolution of ocular motor nerve palsy than anatomic detachment and decompression of the oculomotor nerve from the adjacent and adherent cerebral aneurysm by clipping [1,14]. We agree with their conclusions.

Outcomes for oculomotor nerve palsy after surgical clipping and endovascular coiling have been compared in two study groups respectively. Ahn et al. [11] compared ten coiling patients with seven clipping patients and found no difference in outcome between these two treatment modalities. The predictive factors of recovery after coiling were the severity of initial ocular motor nerve palsy and the interval between onset and treatment. Chen et al. [15] compared six coiling patients with seven surgical clipping patients and found a higher probability of complete oculomotor nerve recovery after surgery. The results suggested that patients with initial partial oculomotor nerve palsy were more prone to complete recovery.

The prognostic factors of oculomotor nerve palsy were reported in terms of aneurysm size, timing of treatment after onset of symptom, degree of palsy, patient's age, coil compaction and recanalization of aneurysm [4,10,11,14,16]. Bhatti et al. [16] suggested that coil compaction and regrowth of the aneurysm was the cause of recurrence. By contrast, Kalish et al. [17] did not consider coil compaction to be related to the recurrence of cranial nerve symptoms. In our study, we found no compaction or recanalization of embolized aneurysms and fistulas upon follow-up cerebral angiography, in any of the cases. This angiographical result may be due to the high packing density of coils upon initial coil embolization.

Two patients had no recovery from symptom after treatment. They had complete oculomotor nerve palsy initially, and the timing of their treatment from onset was later (>20 days). We consider that timing of treatment and severity of symptom may influence the outcomes. It is the reason that a nerve in partial palsy from compression is likely in a neurapraxic phase, a reversible conduction block [14].

In patients with cranial nerve dysfunction who are recovering from an aneurismal mass effect, the resolution of ptosis is usually complete, whereas extraocular muscle function frequently remains impaired [1]. Aberrant regeneration of the ocular motor nerve has been suggested as a possible cause [18]. In our cases, most patients experienced improvement of mydriasis within two days to three months after treatment. Improvement of diplopia took longer and it occurred within 18 months. One patient with partial recovery had residual diplopia only with full recovery from mydriasis and ptosis. Another patient had partial mydriasis recovery.

In our study, eight out of ten patients (80%) were found to have recovered from oculomotor nerve palsy after the coiling of a PComA

aneurysm. We think that this result might be similar or superior to those of prior studies [5,10,11,13,14,17], regardless of treatment modalities. We consider that the high packing density of the coil, which causes complete obstruction of blood into the aneurysm and is followed by the decrease of aneurysmal pulsation, might have affected the outcome.

The present report has some limitations. First, relatively short-terms clinical cases were studied, and angiographic follow-up periods and cases were limited in numbers. Second, the number of cases was too small to analyze statistically. More clinical data with long follow-up are needed to establish the role of endovascular treatment for patients in whom ocular motor nerve palsy is due to posterior communicating artery aneurysm.

Conclusion

Early treatment and initial partial palsy are relevant to improving prognoses. The decrease of aneurysmal pulsation by endovascular coiling may affect early recovery from symptoms. Endovascular treatment is the favored method for treating oculomotor nerve palsy with direct compression or pulsation through intracranial aneurysm.

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